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Multiple Concurrent Sexual Partners, and Sexually Transmitted Disease Dynamics.

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RUNNING HEAD: CONCURRENT PARTNERS AND INFECTION

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ABSTRACT

A straightforward way of modelling concurrent sexual partnerships is proposed, distinct from both the "contact" and "pair-formation" models currently in use. By considering the turnover of both infected and uninfected partners, and employing an approximate measure of risk per infected partner per unit time, the partner-concurrency description may be embedded into a consistent model of sexually transmitted disease dynamics.

The behavior of a very simple example of such a model is explored, and comparisons made with the results for the equivalent instantaneous partnership model (which exists as a limit to the concurrent partnership model). It is shown that if the disease would die out when partnerships are instantaneous, it may be sustainable if enough partners are taken concurrently. By contrast, if the disease would persist with instantaneous partnerships, there is relatively little effect from having concurrent partnerships, unless their number becomes extremely large. The shape of the epidemic curve is shown to be qualitatively different in these two kinds of epidemics.

KEYWORDS: sexually transmitted disease; epidemics; concurrent partners; infection.

1. INTRODUCTION

The recent increase of interest in the transmission dynamics of sexually transmitted diseases (STDs), in large part due to the sexual transmission route for the HIV complex of viruses, has led to a rapid development in the number and sophistication of modelling approaches which are available. Modelling heterogeneity in the rate at which new sexual partners are acquired, the inclusion of a variety of types of structure (sub-groups in the population such as homosexual, bisexual and heterosexual males and females), models which follow pairs of individuals, and the incorporation of arbitrary patterns of infectivity of individuals, over very variable distributed incubation periods, are all now feasible aspects to the investigation of STD dynamics (see Castillo-Chavez 1989 for a review of this literature). In particular, the recent development of mathematical and modelling tools for dealing with any and all population patterns of "mixing" (who has sex with whom), means that general results linking population structure, individual behavior, and disease transmission, are for the first time possible (Busenberg and Castillo-Chavez 1989*a,b*, Castillo-Chavez and Busenberg 1991, Blythe 1991, Blythe *et al.* 1991).

It should be noted however that in almost all studies, sexual partnerships occur in a strictly sequential manner, with no indication of what might happen if individuals can be involved in more than one sexual relationship concurrently. This is true both of the classical contact-distribution models, where partnerships are of infinitesimal duration (*e.g.* Hethcote and Yorke 1984; Anderson *et al.* 1987, 1989, 1990; Sattenspiel 1987*a,b*; Hyman and Stanley 1988; Jacquez *et al.* 1988, 1989; Koopman *et al.* 1988, 1989; Blythe and Anderson 1989; Castillo-Chavez *et al.* 1989*b*), and of pair formation/dissolution models, where individuals move between paired and single status (Dietz and Schenzle 1985; Dietz 1988; Dietz and Hadeler 1988; Hadeler 1989; Waldstätter 1989; Castillo-Chavez *et al.* 1990; Blythe *et al.* 1991).

In this paper we present a model where concurrent partnerships occur, in the context of an extremely simplified description of transmission dynamics. There is only one sex, and no heterogeneity in sexual activity levels, inter-group affinity (*c.f.* Blythe *et al.* 1991), or infectivity. The purpose of this exercise is to illustrate how the turnover of concurrent partnerships may be modelled, and to indicate where this effect may be important in STD transmission dynamics.

2. CONCURRENT PARTNERSHIPS

Assume all individuals in the population undergo the same partner acquisition process, with c new partners acquired per unit time, and an exponentially distributed partnership duration with parameter $1/\sigma > 0$, and mortality occuring at an exponential rate with parameter $1/\mu > 0$. Let $\tau \geq 0$ be the "age" of a partnership, *i.e.* time since its initiation. For sufficiently small increment $\Delta\tau > 0$, let $v(\tau,t)\Delta\tau$ be the number of partners of "age" $[\tau,\tau+\Delta\tau)$ at time t, for any individual, so that the process of partner acquisition and loss ("turnover") is described by an equation of the McKendrick form,

$$\frac{\partial \mathrm{v}(\tau,\mathrm{t})}{\partial \mathrm{t}} + \frac{\partial \mathrm{v}(\tau,\mathrm{t})}{\partial \tau} = -[\sigma+\mu]\ \mathrm{v}(\tau,\mathrm{t}),\ \ \mathrm{v}(0,\mathrm{t}) = \mathrm{c} > 0, \qquad \tau > 0,\ \mathrm{t} \geq 0. \tag{1}$$

If we start at the steady-state of this process (*i.e.* individuals have been active, on average, for a sufficient time that early transients can be ignored), then clearly

$$\mathrm{v}(\tau,\mathrm{t}) = \mathrm{c}\ \exp\Big\{-[\sigma+\mu]\tau\Big\}, \text{ all } \tau \text{ and t.} \tag{2}$$

In Section *5* we consider what happens when this strict assumption is relaxed. Now let $w(\tau,t)\Delta\tau$ be the number of *infected* partners of age $[\tau,\ \tau+\Delta\tau)$ at time t, for any individual. Then the turnover process for infected partners is described by a second McKendrick equation,

$$\frac{\partial \mathrm{w}(\tau,\mathrm{t})}{\partial \mathrm{t}} + \frac{\partial \mathrm{w}(\tau,\mathrm{t})}{\partial \tau} = \mathrm{K}(\tau,\mathrm{t}) - [\sigma+\mu]\ \mathrm{w}(\tau,\mathrm{t}), \qquad \tau > 0,\ \mathrm{t} \geq 0, \tag{3}$$

with $w(0,t)$ the rate of acquiring new partners who are infected, and $\mathrm{K}(\tau,t)$ the rate at which current partners become infected at age τ and time t. In a model with no heterogeneity, we may assume

$$\mathrm{w}(0,\mathrm{t}) = \mathrm{c}\,\frac{\mathrm{I}(\mathrm{t})}{\mathrm{T}(\mathrm{t})}, \tag{4}$$

i.e. one group only, sub-divided by infection status: $I(t)$ are infected, and $T(t)$ is the total population. The initial condition $w(\tau,0)$ must be chosen to be consistent both with the above equations, and with those for the disease transmission dynamics, in which the partner-concurrency description will be embedded.

The choice of the infection rate $K(\tau,t)$ is the key to the model construction process, and must, like $w(\tau,0)$, be consistent with the infection process on the population scale (the transmission model in which the concurrent partnership process is embedded), and with the details of the partner turnover processes, *Eqs (1)* and *(3)*.

We will assume that the infection process can be handled as follows. For an uninfected individual with, say, $W(t)$ infected partners at time t, we write

$$\chi(t) \equiv \beta\,[\sigma+\mu]\,W(t) \tag{5}$$

as the risk per unit time of becoming infected. This corresponds to saying that each partnership lasts on average $1/(\sigma+\mu)$, which is given, and that the risk per partnership is β. It is important to note that β is not a rate. Now $K(\tau,t)$, the rate at which uninfected partners become infected, can be written as the product of the individual risk, *Eq (5)*, and the number of *uninfected* partners an individual has, *i.e.*

$$K(\tau,t) \;=\; \beta\,[\sigma+\mu]\,W(t)\left(v(\tau,t)-w(\tau,t)\right). \tag{6}$$

We may now integrate over $\tau \in [0,\infty)$ in *Eq (3)*, and defining

$$W(t) \equiv \int_0^\infty w(\tau,t)\,d\tau \tag{7}$$

as the total number of infected partners each individual has, we obtain

$$\frac{dW(t)}{dt} = c\,\frac{I(t)}{T(t)} + \beta[\sigma+\mu]\,W(t)\left[\int_0^\infty v(\tau,t)\,d\tau - \int_0^\infty w(\tau,t)\,d\tau\right] - [\sigma+\mu]\int_0^\infty w(\tau,t)\,d\tau$$

$$= c\,\frac{I(t)}{T(t)} - \left([\sigma+\mu]-\beta c\right)W(t) - \beta\,[\sigma+\mu]\,W(t)^2\,, \quad W(0)=0. \tag{8}$$

The simple STD transmission model in which this is embedded is then given by *Eq (8)* plus

$$\frac{dS(t)}{dt} = \Lambda - \beta[\sigma+\mu]\, W(t)\, S(t) - \mu\, S(t)$$
$$\frac{dI(t)}{dt} = \beta[\sigma+\mu]\, W(t)\, S(t) - [\gamma+\mu]\, I(t) \qquad (9)$$

with initial conditions $I(0) = \delta > 0$, $S(0) = \Lambda/\mu$ (*i.e.* the infection is introduced by a small number of infected people entering the population, previously at equilibrium). $\Lambda > 0$ is the rate of recruitment of sexually active individuals (and note that the assumption of steady-state behavior in the partner turnover process *Eq (1)* implies that these "new" recruits have reached their long-term pattern of behavior before becoming at risk; we return to this point in Section *5*), so that Λ/μ is the population size in the absence of the infection. $\gamma > 0$ is the rate at which infected individuals are removed from the active population due to the disease. The initial condition $W(0)$ may conveniently be set to zero (*i.e.* $w(\tau,0) = 0$, all τ), reflecting the assumption that the infection is introduced into the population; if $S(0)$ were of the form $\Lambda/\mu - \delta$, *i.e.* some infecteds are initially part of the population, we are begging the question of their history, implying that $w(\tau,0)$ must be specified in detail as a function of τ. Note also that the form of the infection term in *Eq (9)* must be consistent with that in *Eqs (4)* and *(5)*.

To see how this model, with finite σ, relates to the instantaneous partnership duration model, where $\sigma \to +\infty$, consider the risk function per individual, $\chi(t)$ of *Eq (5)*. From *Eq (8)*, we may write

$$\frac{d\chi(t)}{dt} = \beta c\, [\sigma+\mu]\, \frac{I(t)}{T(t)} + \Big(\beta c - [\sigma+\mu]\Big)\, \chi(t) - \chi(t)^2 . \qquad (10)$$

When σ is very large, we may use time-scaling arguments to approximate *Eq (10)* by the system

$$\frac{d\chi(t)}{dt} \simeq \beta c\, [\sigma+\mu]\, \frac{I(t)}{T(t)} - [\sigma+\mu]\, \chi(t) \simeq 0, \qquad (11)$$

so that we may write, heuristically, that

$$\lim_{\sigma\to\infty} \Big\{\chi(t)\Big\} = \beta c\, \frac{I(t)}{T(t)}, \qquad (12)$$

which is the expected result for instantaneous-duration partnerships (*c.f.* Anderson *et al.* 1987).

3. EQUILIBRIA AND LOCAL STABILITY

The system *Eqs (8)* and *(9)* has the trivial (disease-free) equilibrium $(S^*, I^*, W^*) = (\Lambda/\mu, 0, 0)$, for which the characteristic equation for locally linear perturbations is

$$(\lambda+\mu)\left(\lambda^2+\alpha_1\lambda+\alpha_0\right) = 0\,, \tag{13}$$

where

$$\alpha_1 \equiv (\sigma+\mu)+(\gamma+\mu-\beta c)$$

and

$$\alpha_0 \equiv (\gamma+\mu)\,(\sigma+\mu-\beta c) - \beta c\,(\sigma+\mu)\,.$$

For local stability of $(\Lambda/\mu, 0, 0)$ we require $\alpha_0 > 0$ and $\alpha_1 > 0$, from which we have that

$$R_o \equiv \frac{\beta c}{\gamma+\mu} + \frac{\beta c}{\sigma+\mu} < 1\,. \tag{14}$$

is necessary and sufficient. Note from *Eq (14)* that R_o is exactly the sum of the result for the instantaneous partnership-duration case ($\sigma \to \infty$), and an equivalent term for finite σ. Also note that because $\mu \neq 0$, the value of R_o is bounded as $\sigma \to 0$.

To evaluate the endemic equilibrium, we must solve for (S^*, I^*, W^*) in the equations

$$\Lambda - \beta\,[\sigma+\mu]\,W^*\,S^* - \mu\,S^* = 0 \tag{15a}$$

$$\beta\,[\sigma+\mu]\,W^*\,S^* - [\gamma+\mu]\,I^* = 0 \tag{15b}$$

$$\beta[\sigma+\mu]\,{W^*}^2 + ([\sigma+\mu]-\beta c)\,W^* - c\,\frac{I^*}{T^*} = 0\,. \tag{15c}$$

First note that

$$\frac{I^*}{T^*} = \frac{I^*}{S^*+I^*} = \left(1+\frac{S^*}{I^*}\right)^{-1} = \left(1+\frac{\gamma+\mu}{\sigma+\mu}\,\frac{1}{\beta\,W^*}\right)^{-1}, \tag{16}$$

using *Eq (15a)*. Then defining

$$\rho_1 \equiv \frac{\beta c}{\sigma+\mu} \ , \qquad \rho_2 \equiv \frac{\beta c}{\gamma+\mu} \ , \qquad \hat{\Omega} \equiv \beta \ W^*/\rho_1 = \chi^*/c \ , \tag{17}$$

for $\rho_1 > 0$ (the case $\rho_1 = 0$ was considered in *Eq (12)*), we may re-write *Eq (15c)* as

$$\hat{\Omega}^2 + \left(\frac{\rho_1+\rho_2}{\rho_1\rho_2} - 1\right)\hat{\Omega} - \frac{\left(\rho_1+\rho_2-1\right)}{\rho_1\rho_2} = 0. \tag{18}$$

If $R_o = \rho_1 + \rho_2 > 1$, *Eq (18)* has one real positive solution,

$$\Omega \equiv \frac{1}{2}\left[\left(1 - \frac{(\rho_1+\rho_2)}{\rho_1\rho_2}\right) + \left\{\left(1 - \frac{(\rho_1+\rho_2)}{\rho_1\rho_2}\right)^2 + 4\,\frac{(\rho_1+\rho_2-1)}{\rho_1\rho_2}\right\}^{1/2}\right], \tag{19}$$

from which the (S^*, I^*, W^*) may be calculated. By definition, $0 < W \leq c/(\sigma+\mu)$ (total number of infected partners must not exceed the total number of concurrent partners), so we require $0 < \Omega \leq 1$. From *Eq (19)* we may readily show that $\Omega \leq 1$ requires $\rho_1\rho_2 \geq 0$, and that $\Omega > 0$ requires $\rho_1 + \rho_2 > 1$. The first condition is satisfied by non-negativity of the parameters $(\beta,c,\gamma,\sigma,\mu)$, and the second by $R_o > 1$, so that W^* always satisfies the given constraint. Some special cases of *Eq (19)* may be noted:

$\underline{\rho_1 = 1}$ Then $\Omega = \dfrac{\sqrt{5}-1}{2\rho_2}$,

$\underline{\rho_2 = 1}$ Then $\Omega = \dfrac{\sqrt{1+4\rho_1^2} - 1}{2\rho_1}$,

$\underline{\rho_1 = \rho_2 \equiv \rho}$ Then $\Omega = \dfrac{1}{2\rho}\left(\rho - 2 + \sqrt{8-4\rho+\rho^2}\right)$,

$\underline{\rho_1 \text{ and/or } \rho_2 \text{ large}}$ Then $\Omega \to 1$.

In general, for Ω the solution of *Eq (19)*, we have steady-state values given by

$$(S^*, I^*, W^*) = \left(\frac{\Lambda}{\mu+\rho_1\Omega} \ , \ \frac{\Lambda}{\gamma+\mu}\frac{\rho_1\Omega}{\mu+\rho_1\Omega} \ , \ \frac{\rho_1\Omega}{\beta} \right). \qquad (20)$$

Note in particular that if ρ_1 and ρ_2 both become very large, we have

$$(S^*, I^*, W^*) \to \left(0 \ , \ \frac{\Lambda}{\gamma+\mu} \ , \ \frac{c}{\sigma+\mu} \right),$$

i.e. all individuals in the population become infected, and of course all partners are infected. In *Fig (1)* we illustrate the relationship between ρ_1, ρ_2 and Ω. Below the line $\rho_1 + \rho_2 = 1$ ($\Omega = 0$) there is no endemic steady-state, while above this line the contours of Ω move rapidly towards ρ_1 and ρ_2 at infinity, as $\Omega \to 1$. *Fig (2)* is an example ($\rho_2 = 0.5$) of how the steady-state values *Eq (20)* vary with σ (in this case with $\rho_1 \geq 0.5$). As ρ_1 increases above the critical value of 0.5, S^* rapidly decreases, and I^* rapidly decreases; for larger ρ_1, the changes in S^* and I^* are more gradual. The number of infected partners, W^*, increases smoothly towards its maximum value of $c/(\sigma+\mu)$, *i.e.* $\Omega = 1$, as ρ_1 increases.

Linearising *Eqs (8)* and *(9)* around the steady-state *Eq (20)*, we obtain the characteristic equation (with a factor $[\sigma+\mu]$ absorbed into the time-scale),

$$\lambda^3 + a_2\lambda^2 + a_1\lambda + a_0 = 0,$$

where

$$a_2 = 1 + \frac{\mu}{\sigma+\mu} + \rho_1 \left\{ 3\Omega + \frac{1-\rho_2}{\rho_2} \right\}, \qquad (21)$$

$$a_1 = \left\{ \rho_1\Omega + \frac{\mu}{\sigma+\mu} \right\} \left[1 + \rho_1 \left\{ 2\Omega + \frac{1-\rho_2}{\rho_2} \right\} \right] + \frac{\rho_1}{\rho_2} \left\{ 1 + \rho_1(\Omega - 1) \right\} - \rho_1 \left(1 + \frac{\rho_1\Omega}{\gamma+\mu} \right)^{-1}, \qquad (22)$$

and

$$a_0 = \frac{\rho_1}{\rho_2}\left\{1+\rho_1(2\Omega-1)\right\}\left[\rho_1\Omega+\frac{\mu}{\sigma+\mu}\right] - \rho_1\frac{\left[\frac{\rho_1}{\rho_2}\left(1+\frac{\rho_1\Omega}{\gamma+\mu}\right)-\frac{\gamma}{\sigma+\mu}\right]}{\left(1+\frac{\rho_1\Omega}{\gamma+\mu}\right)^2}. \qquad (23)$$

For local stability of the equilibria *Eq (12)*, we require

$$a_0 > 0\ ,\ a_2 > 0\ ,\ a_1\, a_2 > a_0\ , \qquad (24)$$

none of which are obvious, due to the appearance of Ω. Evaluating *Eq (24)* numerically for a large range of parameter values with $R_o > 1$ failed to find any cases where local stability of the endemic steady state did not hold. It would seem that, as with many HIV/AIDS models, *Eqs (8)* and *(9)* have a very stable pattern of behavior (not all STD models do: there is a growing recognition that infection-related events near the start of the infected period may have a disproportionate effect on the dynamics of the disease; see Huang *et al.* 1990).

4. CONCURRENT PARTNERSHIPS AND THE EPIDEMIC CURVE

Although *Eqs (8)* and *(9)* do not exhibit unstable dynamics for any of the parameter values we tried, it is of interest to see how the average partnership duration $1/\sigma$ affects the shape and timing of the epidemic curve. It is convenient to use scaled variables for this investigation. As we wish to study the impact of σ, we scale using the other parameters, and define

$$X \equiv \mu\, S/\Lambda\ ,\ Y \equiv \mu\, I/\Lambda,\ \ Z \equiv W \Big/ \frac{c}{\gamma+\mu}\ ,\ \text{and}\ \theta \equiv t\,(\gamma+\mu)$$

Then the scaled variable version of *Eqs (8)* and *(9)* may be written

$$\frac{dX(\theta)}{d\theta} = \phi\left(1 - X(\theta)\right) - \rho_2\,\eta\,Z(\theta)\,X(\theta)$$

$$\frac{dY(\theta)}{d\theta} = \rho_2\,\eta\,Z(\theta)\,X(\theta) - Y(\theta) \qquad (25)$$

$$\frac{dZ(\theta)}{d\theta} = \frac{Y(\theta)}{X(\theta)+Y(\theta)} - (\eta - \rho_2)\,Z(\theta) - \rho_2\,\eta\,Z(\theta)^2\,,$$

with initial values $X(0) = 1$, $Y(0) = \epsilon$, $Z(0) = 0$ (where $\epsilon \equiv \mu\delta/\Lambda \ll 1$; we will use $\epsilon = 10^{-3}$), and dimensionless parameter groups

$$\phi \equiv \frac{\mu}{\gamma+\mu}\ ,\ \eta \equiv \frac{\sigma+\mu}{\gamma+\mu} = \frac{\rho_2}{\rho_1}\ .$$

ϕ is the average duration of the infectious period as a fraction of the average life-time; ρ_2 (see *Eqs (14)* and *(17)*) is R_o for the instantaneous-partnership case, *i.e.* the average number of secondary infections an infected person would produce over their infectious life-time, near the start of the epidemic, if partnerships are of infinitesimal duration; η is the ratio of the infected period to average partnership duration (and also equals ρ_2/ρ_1). We take $\mu \simeq 1/30 \simeq 0.0333$ year^{-1}, and $\gamma \simeq 1/10 = 0.1$ year^{-1} for HIV/AIDS, so that $\phi \simeq 0.25$. We choose two values of ρ_2, one less than unity (the epidemic would not be sustainable for instantaneous partnerships), and one greater than unity (epidemic sustainable regardless of σ); arbitrarily, we choose $\rho_2 = 0.8$ and $\rho_2 = 3.0$, respectively. The parameter η may then be varied to assess the impact of concurrent partnerships; note that because of the small but finite death-rate μ, η is constrained to lie in $[\phi, \infty]$. For numerical stability, the scaling used here is appropriate for numerical analysis when σ, and hence η, is "not too large". For $\eta = \infty$ we have the scaled instantaneous partner model,

$$\frac{dX(\theta)}{d\theta} = \phi\left(1 - X(\theta)\right) - \rho_2\,X(\theta)\,\frac{Y(\theta)}{X(\theta)+Y(\theta)} \qquad (26)$$

$$\frac{dY(\theta)}{d\theta} = \rho_2\,X(\theta)\,\frac{Y(\theta)}{X(\theta)+Y(\theta)} - Y(\theta),$$

(*c.f.* Anderson *et al.* 1987) which we shall compare with *Eqs (25).*

$\underline{\rho_2 = 0.8}$ In *Fig (3)* we display the curves of $Y(\theta)/(X(\theta)+Y(\theta))$ (*i.e.* the infected fraction of the total active population) from *Eq (25)* for $\eta = 0.25$, 0.5, 1.0, and 2.0, a sequence where the partnership duration goes from infinity (discounted by deaths) down to about 4.3 years. As η approaches 4.0 ($1/\sigma$ approaches 2.0 years) the epidemic takes an ever-increasing amount of time to appear, while for $\eta > 4.0$ we have $R_o < 1$, so that no epidemic occurs (*Eq (26)* has no endemic equilibrium for this value of ρ_2). This is just a confirmation of the linear analysis of the previous section (see *Eq (14)*), but does serve to underline the point that under conditions where instantaneous partnerships could not sustain an epidemic, finite concurrent partnerships might. In fact it is clear from *Eq (14)* that even if there is no instantaneous infection ($\rho_2 = 0$), but the quantity $\beta c/\gamma > 1$, then a small enough σ can always allow the disease to persist. Note that the shape of the epidemic curve in this case is essentially a rise to a plateau.

$\underline{\rho_2 = 3.0}$ *Fig (4)* shows another set of $Y(\theta)/(X(\theta)+Y(\theta))$ curves for $\eta = 0.25$, 2.5, 10.0, 50.0 (*Eq (25)*), and ∞ (*Eq (26)*, the instantaneous partner case). This sequence has the average partnership duration coming down to about 1.8 months ($\eta = 50.0$). It is clear that there is very little difference between the shape and position of the maxima of the epidemic curves for any η, particularly when η exceeds approximately unity. This occurs because μ is small, so that ρ_1 quickly approaches zero for increasing σ. Note that, in contrast to the small ρ_2 case, the epidemic curve shows a distinct maximum, followed by a decline to the asymptotic value.

These two examples provide a good illustration of the implications of $1/\sigma > 0$ for STD dynamics: if an infection can become endemic purely on the basis of instantaneous partnerships, then the impact of having concurrent partners is small, but if R_o for the instantaneous partnerships case (*i.e.* ρ_2) is less than unity, then an epidemic may occur for average partnership durations that need not be large. Further, the shape of the epidemic is quite different in these cases.

5. NEWLY ACTIVE POPULATIONS

Although the assumption that individuals in the population have achieved their steady-state "turnover" of concurrent partners by the time the epidemic starts is a very reasonable one, it is less clear that the new recruits to the susceptible pool (entry rate Λ year^{-1}) should have this characteristic. We have not yet developed a model formalism which properly considers the gradual process of concurrent partner *accretion* among new recruits, in the context of disease transmission. Such a formalism will have to deal with population dynamic equations more akin to age-structure equations than *Eqs (9)*,the bulk-variable ordinary differential equations for $S(t)$ and $I(t)$. We hope to develop an appropriate formalism in the near future.

As an intermediate exercise, it is worthwhile to consider an STD case intermediate between the simple "steady-state recruits" model of the previous Sections, and one with a full description of recruits' partner accretion. Say the population of interest initially contains only individuals without sexual experience, and that they thereafter accrete partners according to *Eq (1)*. Further, assume that new recruits at any time t have exactly the same characteristic turnover as the population into which they are entering. This approximates the establishment of a new sexually-active population, immigration to which is entirely composed of individuals of similar experience to those already in the population.

In this case we start with $v(\tau,0) = 0$ for all τ, so that we may write the number of concurrent partners at time t as

$$\mathrm{V(t)} \;=\; \int_0^t \mathrm{v}(\tau,\mathrm{t})\,\mathrm{d}\tau \;=\; \mathrm{c}\int_0^t \exp\Big\{-(\sigma+\mu)\tau\Big\}\,\mathrm{d}\tau$$

$$= \frac{\mathrm{c}}{\sigma+\mu}\left(1-\exp\Big\{-(\sigma+\mu)\;\mathrm{t}\Big\}\right), \qquad (27)$$

i.e.,

$$\frac{\mathrm{dV(t)}}{\mathrm{dt}} \;=\; \mathrm{c}-(\sigma+\mu)\;\mathrm{V(t)}, \quad \mathrm{V(0)}=0. \qquad (28)$$

Then we may easily re-write *Eq (8)* for the number of infected partners, $W(t)$, to get

$$\frac{dW(t)}{dt} = c\,\frac{I(t)}{T(t)} - [\sigma+\mu]\,W(t) + \beta\,[\sigma+\mu]\,W(t)\Big(V(t)-W(t)\Big), \quad W(0)=0. \qquad (29)$$

Note that the new system of *Eqs (29)* and *(9)* will have exactly the same $t \to \infty$ endemic steady-state as the original system, *Eqs (8)* and *(9)*, with an extra term for V^*:

$$(S^*, I^*, W^*, V^*) = \left(\frac{\Lambda}{\mu+\rho_1\Omega}\ ,\ \frac{\Lambda}{\gamma+\mu}\,\frac{\rho_1\Omega}{\mu+\rho_1\Omega}\ ,\ \frac{\rho_1\Omega}{\beta}\ ,\ \frac{c}{\sigma+\mu}\right), \qquad (30)$$

and that the only other steady-state is

$$(S^*, I^*, W^*, V^*) = \left(\frac{\Lambda}{\mu}\ ,0\ ,0\ ,\frac{c}{\sigma+\mu}\right), \qquad (31)$$

so that at $t=0$ we are not starting at a steady-state. As *Eq (28)* may be solved independently of the rest of the system, and as $V(t) \to V^* = c/[\sigma+\mu]$ as $t \to \infty$ always represents the asymptotic behavior, we see that the inclusion of *Eq (28)* does not alter the stability properties of the system. As before, for $R_o = \rho_1+\rho_2 < 1$, the disease-free state (*Eq (31)*) is stable, while for $R_o > 1$ the endemic state (*Eq (30)*) exists and is stable. For $R_o < 1$, all that we expect to see is the approach of the partner acquisition process to the equilibrium V^*, with the number of infecteds $I(t)$, and the number of infected partners $W(t)$ going asymptotically to zero.

The only question of interest is whether the trajectories for the "newly active" model are greatly different form those for the original model starting at the steady-state of the acquisition process. Using the same scalings as for *Eqs (25)*, and defining

$$Q \equiv V \Big/ \frac{c}{\gamma+\mu},$$

we have the modified system of dimensionless variables,

$$\frac{dX(\theta)}{d\theta} = \phi\left(1-X(\theta)\right) - \rho_2\,\eta\,Z(\theta)\,X(\theta)$$

$$\frac{dY(\theta)}{d\theta} = \rho_2\,\eta\,Z(\theta)\,X(\theta) - Y(\theta) \qquad (32)$$

$$\frac{dZ(\theta)}{d\theta} = \frac{Y(\theta)}{X(\theta)+Y(\theta)} - \eta\,Z(\theta) + \rho_2\eta\,Z(\theta)\left(Q(\theta)-Z(\theta)\right),$$

$$\frac{dQ(\theta)}{d\theta} = 1-\eta\,Q(\theta).$$

As might be expected, the systems *(25)* and *(32)* show significant differences in the epidemic behavior only when η is small, *i.e.* when partnerships are long, so that many of them can occur concurrently. For example, for the $\rho_2 = 0.8$ case of *Fig (3)*, by the time η exceeds about 2.0, the behavior of $Y(\theta)/(X(\theta)+Y(\theta))$ from *Eqs (25)* and *(32)* are graphicially the same. For the $\rho_2 = 3.0$ case of *Fig (4)*, differences are visible until η exceeds about 5.0. Other numerical examples suggest that this is representative.

6. CONCLUSIONS

The results presented in the previous sections might seem to have some rather bizarre implications for control strategies of STDs. From *Eq (14)* we see that R_o could be written in the form $\beta c D$, where D is essentially the sum of the average partnership duration and the average infectious period. Superficially, this could be interpreted as saying that the practice of having longer-term relationships leads to an *increased* risk of an epidemic. However, the issue here is not one of partnership durations, but rather of having concurrent partners. It is better to think of R_o as the sum of the risk to "stock" (the average number of concurrent partners, $n_S \equiv c/[\sigma+\mu]$) and to "turnover" (the total number of new partners exposed to infection during the infectious period, $n_T \equiv c/[\gamma+\mu]$), *i.e.* $R_o = \beta(n_S + n_T)$, giving the expected number of secondary infections. With this interpretation, it is clear that control

should still be based upon reducing the number of partners, and the risk per partner.

The main model presented here, *Eqs (8) – (9)*, is of extreme simplicity when set against the known features of sexual mixing, and the complications of HIV/AIDS transmission dynamics. Its purpose is twofold. First, it illustrates how an approximation to partner concurrency may be constructed from an elementary consideration of the processes of "turnover" and infection. Second, it demonstrates that the assumption of instantaneous partnerships need not always be a disastrous one, at least with respect to developing a qualitative understanding of transmission dynamics (particularly when data are as scarce and problematical as they are in the study of HIV/AIDS transmission dynamics). If ρ_2 can be calculated for the instantaneous partner case, and is found to be greater than unity, then the incorporation of multiple concurrent partnerships adds little. However, if ρ_2 is less than, and particularly if it is close to, unity, then it may be important to consider the number and duration of concurrent partners, as the epidemic may be capable of persistence, and the epidemic curve may be of the unusual form illustrated in *Fig (3)*. The main implication is that, if ρ_2 is close to unity, then data on the timing and duration of partnerships need to be collected, and not just information on partnership initiation events per unit time.

The alternative model, *Eqs (30)*, approximating an epidemic in a newly sexually active population, suggests that if partnership durations are long, *i.e.* individuals have many concurrent partners, then the process of gaining sexual experience, among members of the population, may also be important. Here the value of R_0 does not appear to be effected, but the detailed shape of the epidemic curve may well be, with potentially large differences in timing. It seems therefore all the more important that data on partner concurrency be obtained.

Extensions of the methods described in this paper to more realistic and complicated model formulations seem possible. For example, we may consider heterogeneity in both activity and partner-concurrency in a population, or incorporate the effects of individual infectivity variations (with time since infection). We are at present exploring some of these possibilities. In particular, we are examining the relationships among models with infinitesimal partnership durations, those with concurrent partnerships, and those where explicit pair formation/dissolution is incorporated (Blythe and Castillo-Chavez, in preparation). It is also possible to extend the partial differential equation formalism of Eqs (1) and (3) to deal with more realistic forms of the partner turnover process (Castillo-Chavez, in preparation). We hope to report on these explorations in the near future.

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FIGURE CAPTIONS

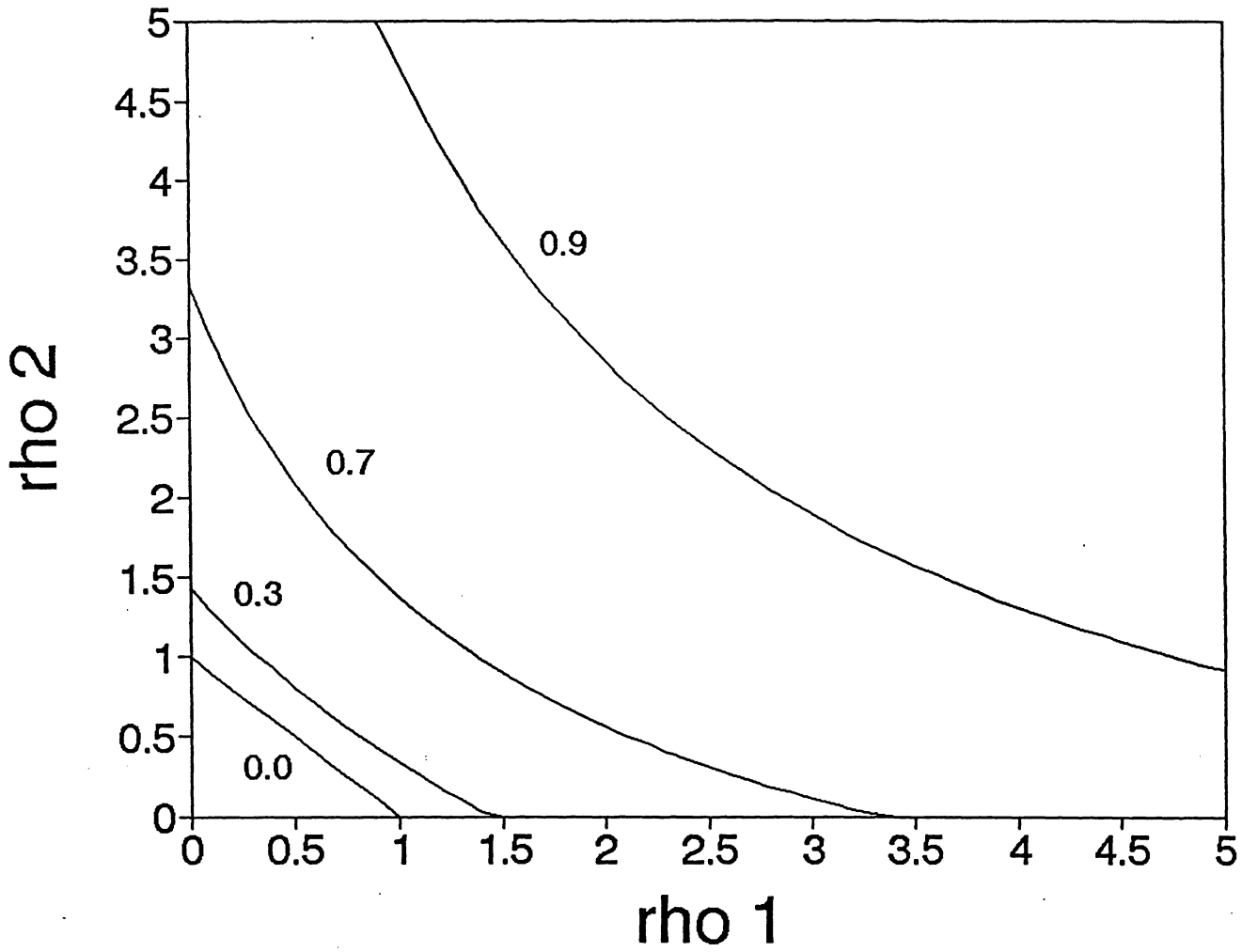
Fig 1. Contours of constant Ω on the (ρ_1, ρ_2) plane, solutions of *Eq (19)* for the steady-state. Below the line $\rho_1 + \rho_2 = 1$ there is no endemic steady state. Contours shown are for $\Omega = 0.0$, 0.3, 0.7 and 0.9. As $\Omega \to +\infty$, contours move rapidly towards infinity. Note that Ω is $W^* \Big/ [c/(\sigma+\mu)]$.

Fig 2. Example of how the steady-state values (S^*, I^*, W^*) of *Eq (20)* vary with σ. Plotted are:- *(a)* $\mu S^*/\Lambda$, *(b)* $\mu I^*/\Lambda$, and *(c)* $\Omega = W^* \Big/ [c/(\sigma+\mu)]$, against ρ_1. Here $\rho_2 = 0.5$, $\mu = 1/30$ year^{-1}, and $\gamma = 1/10$ year^{-1}.

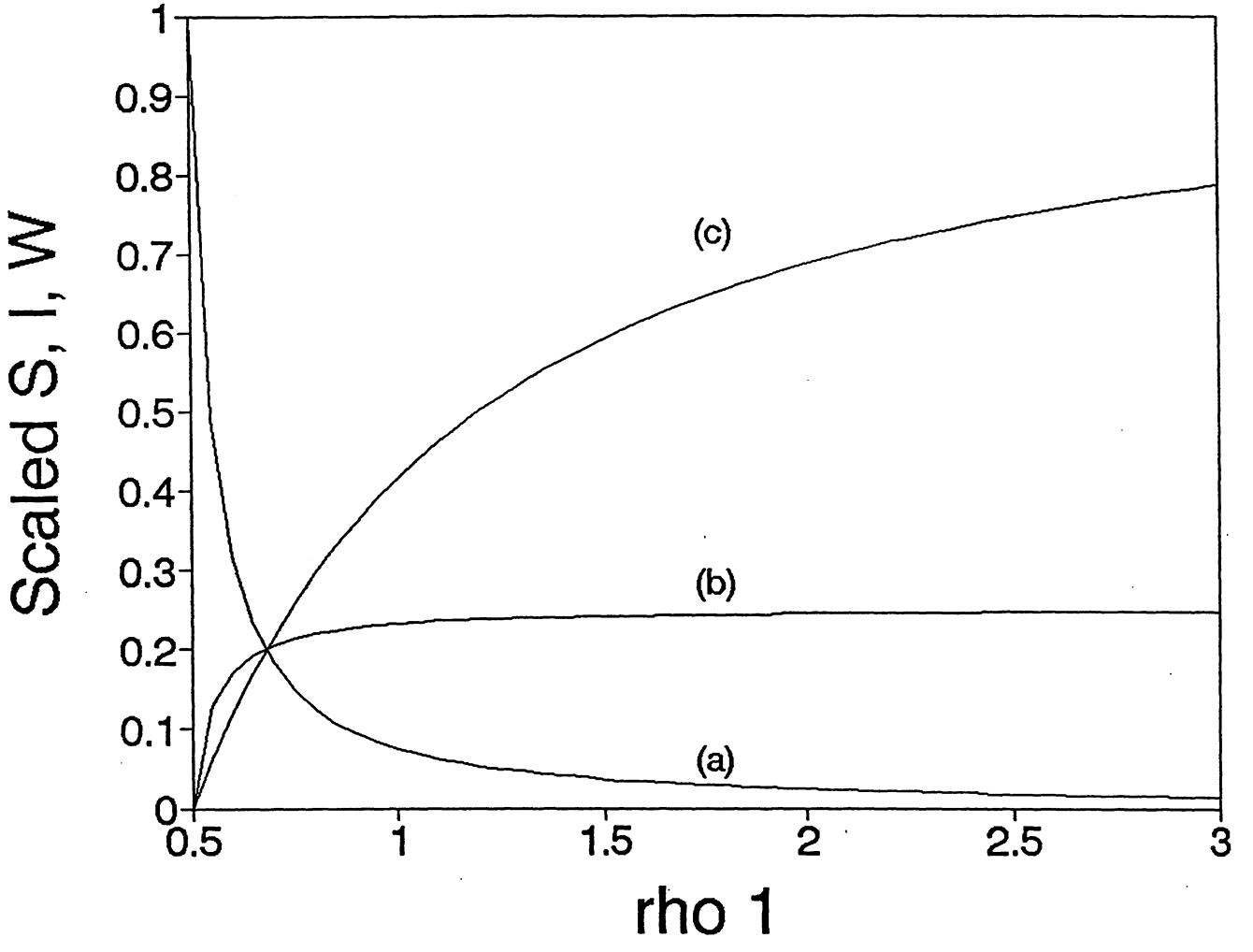
Fig 3. Behavior of *Eqs (25)* for $\rho_2 = 0.8$, and $\eta = 0.25$, 0.5, 1.0 and 2.0; $\eta = 0.25$ is the minimum possible, corresponding to partnerships whose duration is limited only by mortality. Graph shows $Y(\theta)/(X(\theta) + Y(\theta))$, *i.e.* the fraction of the total active population at scaled time θ who are infected. As η approaches 4.0 the epidemic takes progressively longer to appear, and is smaller. Beyond $\eta = 4.0$, no epidemic occurs.

Fig 4. Same as *Fig (3)*, with $\rho_2 = 3.0$, and $\eta = 0.25$, 2.5, 10.0, 50.0, and ∞ (the instantaneous partner case). Curves are too close to label, but at the steady-state (large scaled time) end, η increases from the top curve down. The aberrent curve near the start of the epidemic is the minimal $\eta = 0.25$ case.

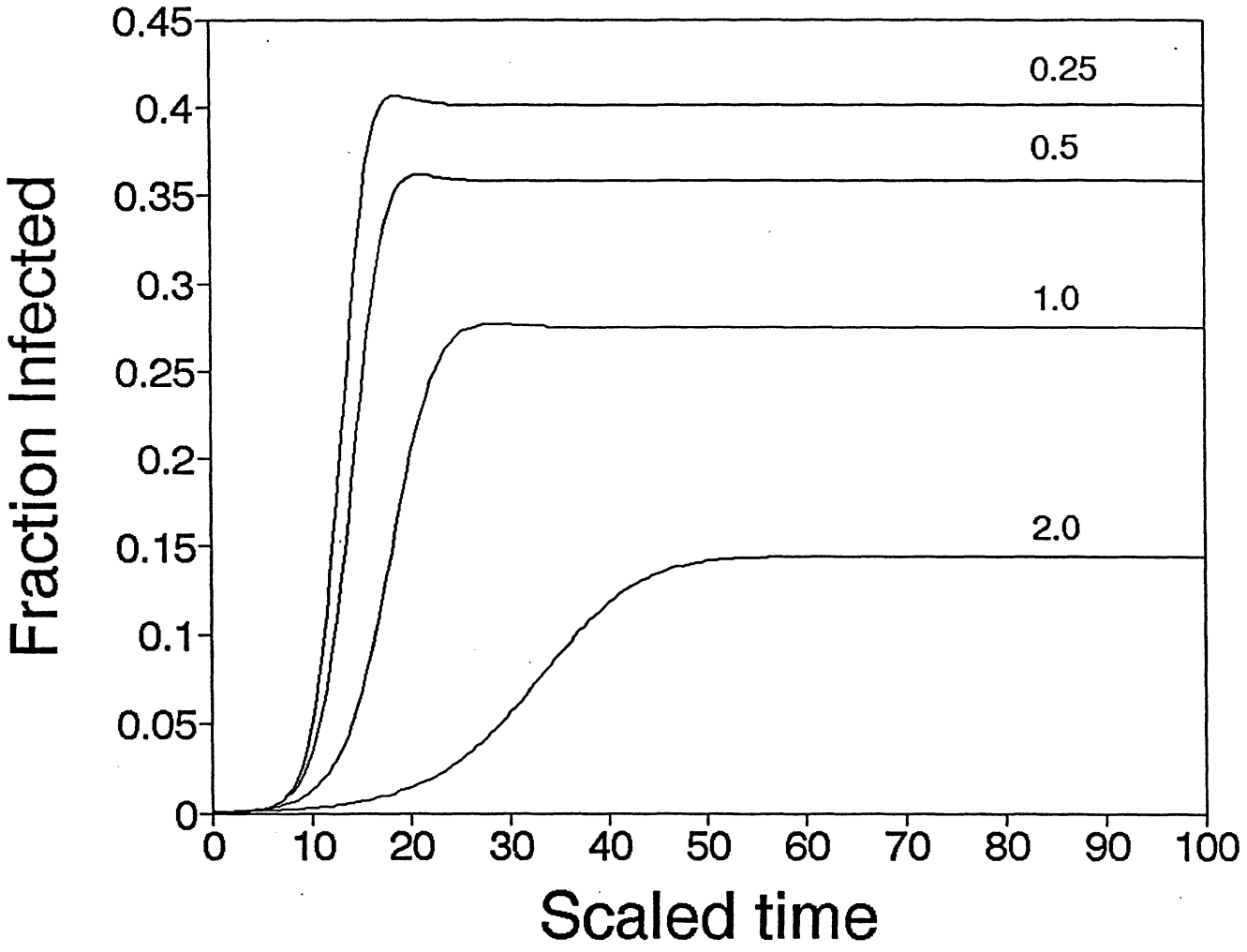
Blythe Fig 1



Blythe Fig 2



Blythe Fig 3


Fraction Infected
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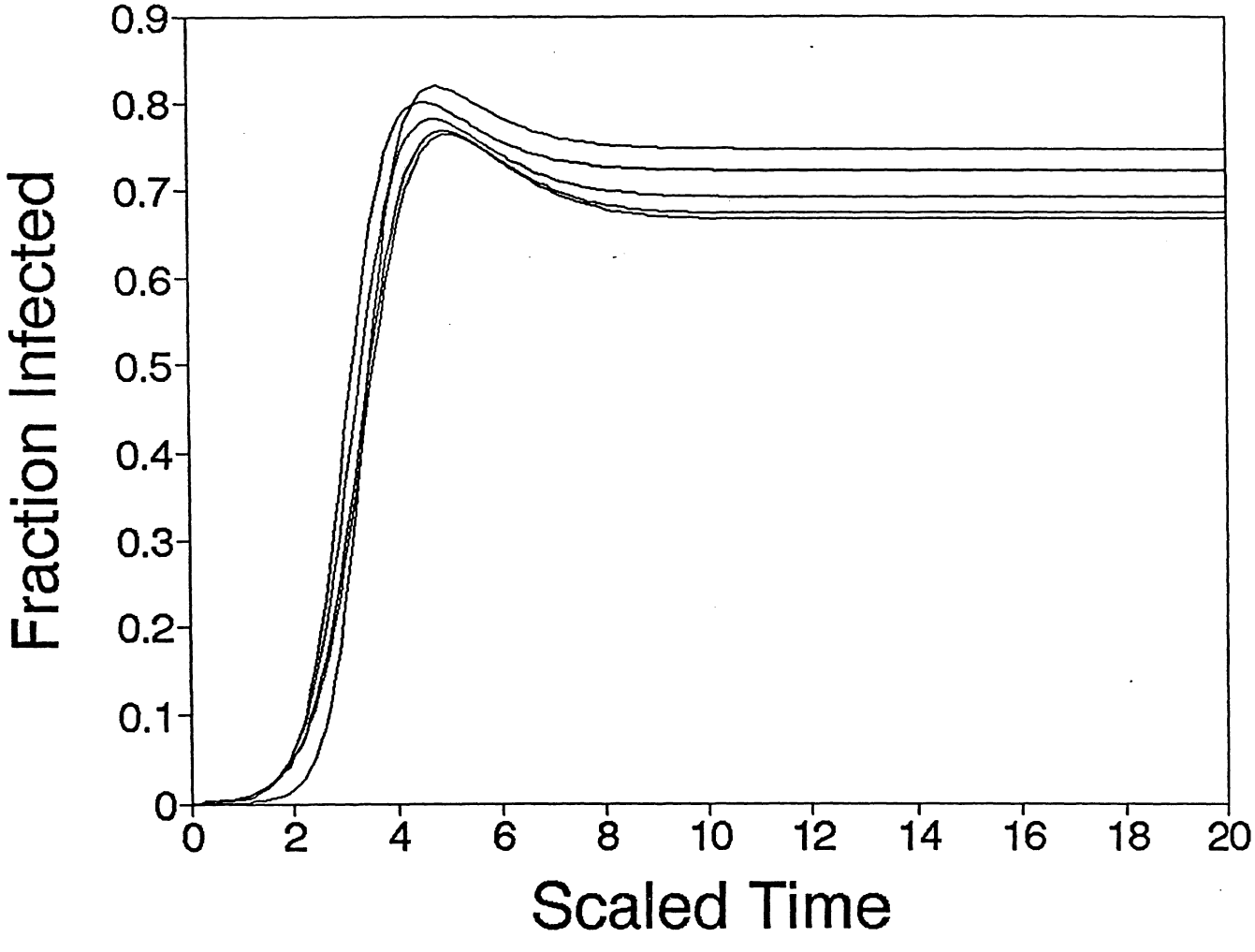
0.35
0.3
0.25
0.2
0.15
0.1
0.05
0
0.25
0.5
1.0
2.0
0
10
20
30
40
50
60
70
80
90
100
Scaled time

Blythe Fig 4


Fraction Infected
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0.8
0.7
0.6
0.5
0.4
0.3
0.2
0.1
0
0
2
4
6
8
10
12
14
16
18
20
Scaled Time